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Original Research Article

MORPHOLOGY AND HISTOPATHOLOGY OF PLACENTA IN IUGR PATIENTS

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ABSTRACT

Background: Fetal growth restriction (FGR) is a failure to achieve the growth potential of a fetus that is promised by the genetic constitution and environmental influences endogenous to the pregnancy. Fetal growth retardation is the second leading cause of perinatal morbidity and mortality, followed only by prematurity. Ultrasound imaging, and in particular color Doppler imaging, allows the study of both the umbilico-placental and uteroplacental circulations. Infarction and intervillous fibrinoid deposition were higher in FGR placenta. The objective is to find out the morphological changes in the placenta of pregnancies with fetal growth restriction. To determine the histopathological changes in the placenta of pregnancies with fetal growth restriction.

Materials and Methods: A cross sectional was conducted at Department of Obstetrics and Gynaecology, Vijay Marie Hospital and Educational Society on 75 pregnant women with intrauterine growth restriction during September 2019 to August 2020. Patients with gestational age from 32 to 42 weeks who are diagnosed with FGR, singleton Pregnancy were included in this study. Multiple pregnancies, polyhydramnios and patients with unsure dates and irregular menstrual cycles were excluded.

Results: Majority of the patients belong to the age group of 21-25 years. High resistance in Umbilical artery doppler is seen in 25.3% patients. Low resistance in Middle cerebral artery doppler is seen in 6.7% patients. Notching in uterine artery is present in 6.7% patients. 53.3% patients had Calcifications and 20% had hemorrhage/ infarct in placenta. Histopathological findings revealed that Peri villous fibrin deposition is present in 97.3% patients.

Conclusion: There is predominant role of placental causes in cases of idiopathic fetal growth restriction.

Keywords: Placenta, Ultrasound, Histopathology, Fetal growth restriction, Doppler.

INTRODUCTION

Fetal growth restriction (FGR) is a failure to achieve the growth potential of a fetus that is promised by the genetic constitution and environmental influences endogenous to the pregnancy. Fetal growth retardation is the second leading cause of perinatal morbidity and mortality, followed only by prematurity.[1] The infant born with FGR is recognized as having an increased risk of in utero mortality, neonatal morbidity and mortality and as well as long term neurological complications. The neonatal mortality rate for FGR infants born at 38 weeks was 1% compared with that of 0.2% in those with appropriate birth weights.[2]

IUGR or Fetal growth restriction (FGR) is defined as the inability of a fetus to maintain expected growth, with estimated fetal weight or actual birth weight below the 10th percentile for gestational age with a pathologic restriction of fetal growth.[3] Placental factors and hypoxemia are keys to FGR and fetal death. It is a condition associated with placental insufficiency. Conditions resulting in placental

dysfunction may be recurrent. Any abnormality in placenta leads to direct insult to the fetus in-utero.[4] Ultrasound is the most widely used, standard and simple way of detecting and confirming FGR. Sonographic parameters both conventional and Doppler USG are used for diagnosis of FGR. Ultrasound imaging, and in particular color Doppler imaging, allows the study of both the umbilico-placental and uteroplacental circulations. These techniques have been used extensively in the screening of placental-related complications of pregnancy and the management of a fetus presenting with primary or secondary FGR.[5]

In a histopathological evaluation of placenta in FGR pregnancies, the weight of FGR placenta was less than normal placenta. Infarction and intervillous fibrinoid deposition were higher in FGR placenta. In addition thickening of basal membrane and cytotrophoblastic hyperplasia were more common among FGR placenta. All the main histopathological findings pointed to placental blood flow reduction and fetal blood flow restriction.[6]

Objectives

1. To find out the morphological changes in the placenta of pregnancies with fetal growth restriction.
2. To determine the histopathological changes in the placenta of pregnancies with fetal growth restriction.
3. To determine the association between morphological and histopathological changes in the placenta in relation to fetal outcome.

MATERIALS AND METHODS

A cross sectional was conducted at Department of Obstetrics and Gynaecology, Vijay Marie Hospital and Educational Society on 75 pregnant women with intrauterine growth restriction during September 2019 to August 2020. Sample size was calculated from the data available from the National Health Portal7, which was published in 2018 showing prevalence of intra uterine growth retardation as 24%. All Pregnant women between the ages 18 to 35 years, patients with gestational age from 32 to 42 weeks who are diagnosed with FGR, singleton Pregnancy, patients with minimum four antenatal checkups were included in this study. Multiple pregnancies, polyhydramnios and patients with unsure dates and irregular menstrual cycles were excluded.

Upon IUGR suspicion based on the following parameters i.e., gestational age, and series of ultrasound from 28week onwards Doppler sonography were done for umbilical artery uterine and middle cerebral arteries resistance to confirm the diagnosis. After delivery, all the necessary data regarding, mother, newborn and morphologic assessment of placenta i.e. placental weight, cord insertion and number of the umbilical vessels, embryonic membranes, colour of the maternal and fetal surfaces of placenta, and the probable haemorrhages and infarcts was collected and recorded using a predesigned proforma. The placenta is kept in formalin 10% solution and sent to the laboratory, where it was examined by the pathologist. Data entry was done using M.S. Excel and statistically analyzed using Statistical package for social sciences (SPSS Version 16) for M.S Windows.

RESULTS

Majority of the patients belong to the age group of 21-25 years (66.7%), followed by 26-30 years (21.3%), <20 years (6.7%) and >30 years (5.3%). 58.7% patients were primi, 22.7% patients belong to 2nd gravida, 16% patients belong to 3rd gravida and 2.7% patients belong to 4th gravida. Gestational age is 28-30 weeks in 13.3% patients, 25-37 weeks in 34.7% patients and 38-42 weeks in 52% patients. Mode of delivery is vaginal delivery in 44% patients and LSCS in 56% patients. Majority of the patients belongs to middle class (52%) followed by lower middle class (26.7%) and upper middle class (21.3%). 6.7% patients are underweight, 20% patients are overweight and 10.7% patients are obese. Majority of the patients had no associated risk factors, however anemia is present in 2.7% patients, Gestational Diabetes Mellitus is present in 4.0% patients, Gestational Hypertension is present in 4.0% patients and Severe Pre-eclampsia is present in 5.3% patients.

In this study, all are live births. Weight of the baby is <1.5 kg in 10.7% patients, 1.5-2.5 kg in 62.6% patients and>2.5kg in 26.7% patients. Majority of the newborn with birth weight ranging between 1.5 to 2 kg got admitted in NICU and discharged subsequently with no gross morbidities. APGAR score at 1 min is 7-10 in 85.3% patients and 4-6 in 14.7% patients. APGAR score at 5 min is 7-10 in 93.3% patients and 4-6 in 6.7% patients.

Table 1: Distribution of patients based on the Disparity on ultrasound

Disparity on ultrasound	Frequency	Percent
<2 Weeks	36	48.0%
2 to 4 Weeks	27	36.0%
>4 Weeks	12	16.0%
Total	75	100.0%

High resistance in Umbilical artery doppler is seen in 25.3% patients. Low resistance in Middle cerebral artery doppler is seen in 6.7% patients. Notching in uterine artery is present in 6.7% patients.

Placental weight is <450gms in 29.3% patients and 450-550gms in 70.7% patients. Thickness of placenta is <35mm in 17.3% patients, 35-45 mm in 81.3% patients and >45 mm in 1.3% patients. Umblical cord

Attachment is Central in 98.7% patients and Velamentous in 1.3% patients. 53.3% patients had Calcifications and 20% had hemorrhages in placenta. Chorioamniotic Membranes are seen in 18.7% patients. Cord is normal in 89.3% patients, true knot cord is seen in 8% patients and hypercoiling cord is seen in 2.7% patients.

Table 2: Distribution of patients based on the Histopathological findings

Histopathological findings	Frequency	Percent
Perivillous fibrin deposition	73	97.3%
Stromal fibrosis	12	16.0%
Cytotrophoblast proliferation	11	14.7%
Villous infarction	19	25.3%
Thickening of the villous trophoblastic basal membrane	8	10.7%
Intervillous thrombi hematoma	36	48.0%
Villitis	27	36.0%
Focal chorangiosis	62	82.7%
Syncitial knots	68	90.7%

Table 3: Distribution of patients based on the various parameters and risk factors

			Risk factors		Total	P Value
			Yes (n=12)	No (n=63)		
Parity Index	Primi	n	5	39	44	0.01
		%	41.7%	61.9%	58.7%	
	Gravida 2	n	3	14	17	
		%	25.0%	22.2%	22.7%	
	Gravida 3	n	2	10	12	
		%	16.7%	15.9%	16.0%	
	Gravida 4	n	2	0	2	
		%	16.7%	0.0%	2.7%	
Umbilical artery doppler	Normal	n	8	46	54	0.67
		%	66.7%	73.0%	72.0%	
	High Resistance	n	4	15	19	
		%	33.3%	23.8%	25.3%	
	Absent	n	0	2	2	
		%	0.0%	3.2%	2.7%	
Middle cerebral artery doppler	Normal	n	10	60	70	0.17
		%	83.3%	95.2%	93.3%	
	Low Resistance	n	2	3	5	
		%	16.7%	4.8%	6.7%	
Placental weight	<450 gms	n	7	15	22	0.02
		%	58.3%	23.8%	29.3%	
	450-550 gms	n	5	48	53	
		%	41.7%	76.2%	70.7%	
Placenta	Normal	n	0	20	20	0.02
		%	0%	31.7%	26.7%	
	Calcifications	n	6	34	40	
		%	50%	54.0%	53.3%	
	Hemorrhage/ infarct	n	6	9	15	
		%	50%	14.3%	20%	
Peri villous fibrin deposition		n	11	62	73	0.29
		%	91.7%	98.4%	97.3%	
Thickening of the villous trophoblastic basal membrane		n	1	7	8	0.62
		%	8.3%	11.1%	10.7%	
Syncitial knots		n	10	58	68	0.31
		%	83.3%	92.1%	90.7%	

DISCUSSION

A normal placental structure and function is required for the development of a normal healthy fetus at the term. However, if there is an abnormality in placental development, then that could hamper fetal growth and wellbeing resulting in severe complication ranging from IUGR to death of fetus.[8-10] IUGR is a common diagnosis seen in obstetrics patients that increases the risk of perinatal mortality and morbidity.[11,12] In most instances, no obvious maternal or fetal cause could be assigned to IUGR, yet gravid mothers present with severe weight loss in fetuses. In such cases, researchers have implicated placenta to be culpable in causing disproportionately low fetal weight resulting in IUGR.[11,13] The placenta is the only vital organ of perinatal life, which can be examined, without hazards to the mother and the baby. However, the placenta provides a paradox as it is one of the most readily available structures for examination, but is one of the least known.[14-16] The present study was conducted to know the various morphologic and histopathologic changes of placenta in IUGR pregnancies.

In this study, Majority of the patients belong to the age group of 21-25 years. In a study done by Gunyeli L et al,[4] & Ghomian N et al,[6] mean age were 26.5 + 6.05 years and 27.6 + 5.3 years respectively. Gestational age is 38-42 weeks in 52% patients. In a study done by Gunyeli L et al,[4] mean gestational age was 33.67 + 4.37 years. In a study done by Ghomian N et al,[6] mean gestational age was 37.7 + 1.9

In this study, all are live births. In a study done by Sawant LD et al,[11] The fetal outcome in IUGR showed that 3.77% of fetuses suffered from Jaundice while 1.89% suffered from Respiratory Distress Syndrome (RDS) and 1.89% had perinatal death approximately 93% fetuses were normal with no anomaly, and TORCH and HIV infections. The favorable outcome in pregnancies related to IUGR was due to good obstetric care, NICU facilities and good health care professional staff in the Department of Obstetrics and Gynecology at our hospital that were well trained to manage such high risk pregnancy and obstetric emergency.

In this study, Weight of the baby is 1.5-2.5 kg in 62.6% patients. In a study done by Ghomian N et al,[6] & Shrivastava N et al,[17] mean birth weight was 2.12 + 0.38 kg and 1.99 ± 0.33 kg respectively. APGAR score at 1 min is 7-10 in 85.3% patients and 4-6 in 14.7% patients. APGAR score at 5 min is 7-10 in 93.3% patients and 4-6 in 6.7% patients. In Park SY et al,[18] APGAR score at 1 min is <7 in 13.3% patients and APGAR score at 5 min is <7 in 6.7% patients.[18]

In this study, Anemia is present in 2.7% patients, Gestational Diabetes Mellitus is present in 4.0% patients, Gestational Hypertension is present in 4.0% patients and Severe Pre-eclampsia is present in 5.3% patients. In a study done by Sawant LD et al,[11] examination of risk factors showed gestational hypertension 13% in growth restricted fetuses while 5.66% in normal fetuses. Even though, gestational hypertension is seen more in IUGR pregnancies compare to normal pregnancies, the data is not statistically significant. In a study done by Sujatha C et al,[19] 40 placentae were collected from IUGR among them 50% placentae were of PIH, 7.5% were of anaemia, 7.5% were of polyhydramnios, 7.5% were of oligohydramnios, 7.5% were due to heart disease & 20% were due to idiopathic reasons.

In this study, Placental weight is <450gms in 29.3% patients and 450-550 gms in 70.7% patients. In a study done by Ghomian N et al6 & Kotgirwar S et al,[20] mean placental weight was 440 + 64.5 gms and 281+24.69gms respectively. In a study done by Sawant LD et al,[11] The placental weight were predicted according to gestational age by ultrasound and compared with actual placental weights in the study group which showed a difference of approximately 70-80 g in predicted placental weight and actual placental weight in grams. It also showed that placental weight is 1/5th of fetal weight. In a study done by Khajuria R et al,[21] Average placental weight in IUGR group was 425 gms. In a study done by Sujatha C et al,[19] Maximum number of IUGR cases had the placental weight within 200 – 300gms. Placental disc thickness is an indirect measure of the extent of development of the nutrient exchange surface of the placenta essential to its successful support of fetal growth. Any abnormal change in the thickness either thin or thick placentae have been correlated to adverse pregnancy outcome140. In this study, Thickness of placenta is <35mm in 17.3% patients, 35-45 mm in 81.3% patients and >45 mm in 1.3% patients. In a study done by Sawant LD et al,[11] the placental thickness was also compared between normal fetuses and IUGR, and it demonstrated that placental thickness is decreased in IUGR.

In this study, Umbilical cord insertion is Central in 98.7% patients and Velamentous in 1.3% patients. In a study done by Ghomian N et al,[6] Umblical cord insertion is Central in 39.1% patients and eccentric in 60.9% patients. In a study done by Sawant LD et al,[11] none of the IUGR placentae showed eccentric or villamentous insertion. In a study done by Khajuria R et al,[21] Umblical cord insertion is Central in 73.3% patients and eccentric in 26.7% patients. In a study done by Bjoro K and Davis BR et al., villamentous and marginal insertion for umbilical cord are more common in IUGR.[22,23]

In this study, 53.3% patients had Calcifications and 20% had hemorrhage/ infarct in placenta. In a study done by Sawant LD et al,[11] increase in placental calcification in IUGR (22 %) compared to normal placenta (19%). Although the placenta from both the groups showed calcification, the placenta from fetal growth restricted pregnancy has dense calcification per cotyledon compare to the placenta from the normal pregnancy.

In this study, Chorioamniotic Membranes are seen in 18.7% patients. In a study done by Ghomian N et al6 & Khajuria R et al,[21] Chorioamniotic Membranes are seen in 4.3% & 6.7% patients respectively. In a study done by Shrivastava N et al,[17] No pathological changes were observed in membranes.

In this study, Cord is normal in 89.3% patients, true knot cord is seen in 8% patients and hypercoiling cord is seen in 2.7% patients. In a study done by Shrivastava N et al,[17] No pathological changes were observed in umbilical cord.

In this study, Histopathological findings revealed that Peri villous fibrin deposition is present in 97.3% patients, Stromal fibrosis is present in 16% patients, Cytotrophoblast proliferation is present in 14.7% patients, Villous infarction is present in 25.3% patients, Thickening of the villous trophoblastic basal membrane is present in 10.7% patients, Intervillous thrombi hematoma is present in 48% patients, Villitis is present in 36% patients, Focal chorangiosis is present in 82.7% patients and Syncitial knots is present in 97.3% patients. In Gunyeli L et al Chorionic villitis occurred in 69% patients, 31% had placental intravascular thrombi, Perivillous fibrin deposition and fibrinoid necrosis was more common (65%). Ghomian N et al showed Microscopic infarction (52.1%), Thrombosis (34.7%), Avascular

villi (30.4%), microscopic tissue ischemia (21.7%), Intervillous fibrinoid deposition of >10% (43.5%), cord vasculitis (4.3%), chorangioma (4.3%) and intravenous hemorrhage (8.6%). In Park SY et al.,18 histologically, IUGR was characterized by increased incidence of decidual vasculopathy (31.1%, $p<0.05$), multiple and severe infarct ($p<0.05$), villous fibrosis (31.1%, $p<0.05$), syncytiotrophoblastic knots (86.7%, $p<0.05$), and higher degree of increased perivillous fibrin deposition ($p<0.05$). In Kotgirwar S et al,[20] The Syncytial knots>30% were found in 60% cases, Fibrinoid necrosis >5% was found in 46.7% cases, Placental infarction>5% was found in 1.8% cases, Perivillous fibrinoid deposition >5% was found in 16.7%, calcification in 60%cases. No pathological changes were observed in umbilical cord and membranes. In Shrivastava N et al,[17] increased syncytial knotting (84.5%) are seen. In the villi, number of areas with more than 5 fibrinoid necrosis and hyalinised areas in IUGR group was noted in 28.5% and 50.5% placentas respectively. In the intervillous space stroma, calcific areas more than 5 was found in 152 patients with IUGR (76%). In Sawant LD et al,[11] showed nonspecific inflammation of placental villi with loss of vascular bed at syncytiotrophoblast layer resulting in ischemic damage to the placenta. Placental ischemia and infarcts were often seen in women with hypertension and with the increase in the severity of toxaemia in pregnancy, all the placental changes are exaggerated. In Kavita M et al,[24] Intervillous fibrin deposition (64%), increased syncytial knotting (64%), stromal fibrosis (65%), cytotrophoblastic hyperplasia (44%) and basement membrane thickening (40%) were seen along with hypovascular villi and infarction were present in 32% and 28% respectively. In Khajuria R et al,[21] Infarction, intervillous thrombosis, chorionic villitis, hemorrhagic endovasculitis, placental intravascular thrombi, perivillous fibrin deposition, fibrinoid necrosis and villous edema were found in IUGR patients. In Sujatha C et al,[19] cytotrophoblastic hyperplasia, abnormal villous vascularity, basement membrane thickening, fetal stem artherosis, fibrinoid necrosis, syncytial knots, calcifications are seen.

CONCLUSION

It can be concluded that study of morphological and histopathological aspects of placenta provides a major insight into the prenatal health of the fetus and mother as it showed fetal growth restriction is more prevalent in patient even without any risk factors with significant reduction in foetoplacental weight and placental dimensions. It draws the attention that there is predominant role of placental causes in cases of idiopathic fetal growth restriction.

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